Traumatic posterior fossa subdural hematoma

Posterior fossa subdural hematoma cases account for approximately 0.2% $^{1)}$ of all head traumas and 0.3-3% $^{2)}$ $^{3)}$ of the traumatic subdural hematomas.

Rarely, subdural hematomas may be spontaneous, with no previous trauma. These cases are usually secondary to bleeding from an underlying pathology such as arteriovenous malformation (AVM), tumor or coagulation dysfunction ^{4) 5)} According to the medical literature, only five cases of acute spontaneous subdural hematomas in the posterior fossa were reported ^{6) 7)}.

The low rate of occurrence of this type of hematoma in the posterior fossa could be explained by the few number of bridging veins in this region and the uncommon direct damage to the occipital dural sinus 8 ⁹.

Moreover, Goldsmith and Plunkett hypothesize that since the posterior fossa space is almost completely fulfilled by the brainstem and cerebellum, the movement of the skull base is synchronic to those structures, therefore no shear forces occur in this compartment ¹⁰.

Case series

Ten patients with traumatic posterior fossa SDHs were admitted to our hospital. There were seven males and three females, with an age range of 3 years to 97 years (mean, 57.5 years). Coagulopathies were observed in five patients. The causes of injury were motor vehicle crash in three patients, falls in six patients, and being hit by an iron plate in one patient. The mean admission Glasgow Coma Scale score was 8.3. Skull fractures were revealed in six patients. Hematoma sizes ranged from 5 mm to 20 mm (mean, 7.7 mm). Two patients presented with isolated posterior fossa SDHs, and eight patients presented with associated intracranial lesions. Only one patient was treated surgically for posterior fossa SDHs associated with intracerebellar hematomas. The poor outcome rate was 90% and the mortality was 50%.

A review of the literature revealed the following characteristics of posterior fossa SDHs: (1) a relatively high frequency of occipital impacts and fractures, (2) a low Glasgow Coma Scale score, (3) a high frequency of associated intracranial lesions, especially supratentorial lesions and intracerebellar hematomas, (4) a potential for lesion evolution, especially within 2 days, and (5) a high poor outcome rate and mortality ¹¹.

1) 6)

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